Influence of temperature dependent vector population growth to the spread characteristics of arbovirus infections

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Abstract— **In this paper, the evolution of dengue cases is studied by explicitly incorporating the influence of temperature on the dynamics of the life cycle of mosquitoes. The resulting proposed model couples the dynamics of humans and vectors (both infected and healthy), allowing for the derivation of closed-form expressions for equilibrium points, stability conditions, and basic reproduction number. Identification of transmission parameters was performed using real data from the Dengue cases in Brazil, achieving an accurate reconstruction of both the number of infected individuals and the oscillatory seasonal trend. The identified model shows a good epidemic reconstruction profile, making possible a prediction of successive sanitary emergency. Nevertheless, it suggests a not negligible influence of other environmental and climate factors in predicting future sanitary emergency, as well as human intervention actions, mainly represented by the increase in the application of test and sanitary surveillance.**

I. INTRODUCTION

As described by the World Health Organization (WHO) [1], Dengue is caused by the Dengue virus (DENV) and transmitted to humans through the bites of infected mosquitoes. This virus has been known for more than two centuries and is particularly prevalent during and after the rainy season in tropical and subtropical regions of Africa, Southeast Asia, China, India, the Middle East, Latin and Central America, Australia, and several areas of the Pacific. In particular, in 2024, there have been more than 12 million cases in Brazil, Argentina, Mexico, Colombia and Paraguay. Mosquitoes breed in small pools of water near homes and are most active during the day; they prefer warm, humid climates, especially after rain. Risk factors include the presence of stagnant water (pots, tires, containers), urbanization, and international travel.

In recent decades, the spread of Dengue has increased across many tropical regions. Climate change and urbanization are favoring its spread even in temperate areas, where there are sporadic autochthonous cases. In particular, in Europe it occurs mainly as an imported disease, with its rise linked to the growing frequency of the movement of goods and people. According to the WHO, the incidence of Dengue has increased 30 times in the last 50 years, and nearly half of the world's population is at risk of infection. For this reason, the application of techniques and methods that allow a better understanding of the evolution of this disease is crucial to analyze and control its spread. Globalization and climate change are significant drivers of the spread of the Dengue virus. An important feature of vector-borne diseases is the influence of climatic factors: favorable climatic and environmental conditions facilitate the spread of the virus even in regions not accustomed to such epidemics [2]. Therefore, it is important to adequately model the dynamics of Dengue considering its spatial and temporal spread; in particular, the periodicity of the epidemic is correlated with local characteristics of temperature, humidity and rainfall [3], [4], [5]. In [6], the problem of periodicity in Dengue spread is modeled by means of time dependent contact rates between human and mosquitoes populations; this allows to describe the different stages of Dengue spread that increases during the warmer and more humid months, followed by a fast decrease in the driest seasons. More recently, in [7] one SEIR model is associated with a human population, while a SEI model describes the mosquitoes one; the interesting peculiarity of this approach is the attention to modifying human behavior related to urbanization as a key role in influencing the spread of the epidemic amid climate change, as well as acting on vector control. Moreover, in the model, a direct impact of temperature is introduced in the life cycle of mosquitoes. An interesting spatio-temporal climate based modeling is proposed in [8], where the relationship between climate and human Dengue incidence is studied referring to Perù over a period of more than a decade, up to 2021. The pursued goal is to understand climatic drivers of Dengue incidence and, therefore, propose suitable containment measures. Also in [9] the significant role of climate aspects in the spread dynamics of Dengue has been stressed. Based on all these considerations, in this paper temperature has been explicitly introduced into the model, as it significantly influences mosquito dynamics. More precisely, the dynamics of mosquitos life cycle is described, considering four stages, eggs, larvae, pupae, and adults, whose dynamics is influenced by temperature changes, by means of transition rates from each stage, as well as the mortality rates. As reference scenario, it is considered the situation in Brazil, particularly severe in the last years. The structure of the paper is as follows. Section 2 presents the formulation of the mathematical model, describing both the human population infection dynamics and the mosquito life cycle. Section 3 is devoted to the qualitative analysis of the model, while Section 4 reports the results of the parameter identification procedure and the corresponding numerical simulations. Conclusions and future works end the paper.

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II. THE MATHEMATICAL MODEL

In this section, a mathematical model is developed to describe the evolution of Dengue, accounting for the interaction between the human population and the mosquito's one. In particular, the effect of temperature on vector dynamics is introduced by modeling the different stages of the mosquito life cycle and integrating these dynamics with the model of healthy and infected humans. The aim is to obtain a mathematical model able of capturing the main mechanisms of transmission and spread of the infection according to climate changes, temperature by now, thereby providing a solid basis for phenomenon analysis and mitigating solutions.

A. Mosquitoes and temperature

To model the evolution of Dengue cases, it is essential to account for the fact that temperature has a strong and direct impact on the cycle of life dynamics of mosquitoes. In this work, the focus is on Dengue cases in Brazil, as it is severely affected with more than 4.6 million cases in 2023 and more than 2400 deaths with a mortality rate that increases dramatically (up to 40%) in the most severe haemorrhagic forms.

For this reason, the mean temperature variations for Brazil were constructed using data from 2014 to 2023. Based on the maximum and minimum temperature values provided by the data set [10], the daily mean temperature was first calculated. The missing daily values were then estimated through linear interpolation to obtain a continuous daily temperature time series. Subsequently, the weekly mean temperatures for the ten-year period were derived, as shown in Fig. 1.

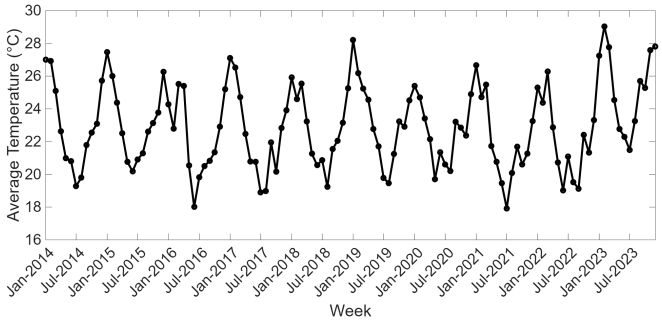


Fig. 1: Weekly average Temperature in Brazil (2014–2023)

The mosquito life cycle considered here consists of four stages: eggs, larvae, pupae, and adults. Life stages are described by the following ordinary differential equations:

$$\dot{E} = \beta(T)A - (m_E(T) + f_E(T))E \tag{1}$$

$$\dot{L} = f_E(T)E - \left[m_L(T)\left(1 + \frac{L}{K_L}\right) + f_L(T)\right]L \tag{2}$$

$$\dot{P} = f_L(T)L - (m_P(T) + f_P(T))P \tag{3}$$

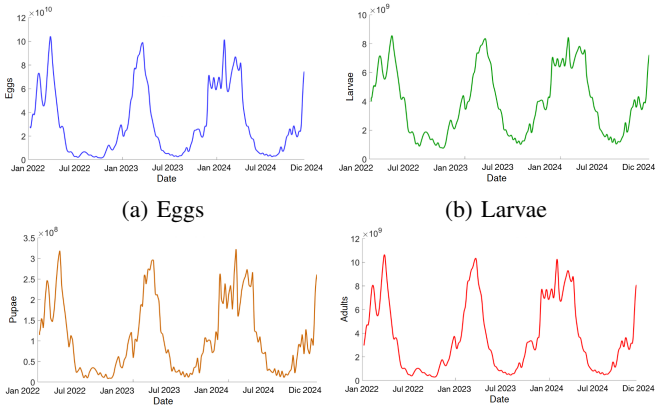
$$\dot{A} = \sigma f_P(T)P - m_A(T)A \tag{4}$$

where: E is the number of eggs, L the number of larvae, P the number of pupae, A the number of adult females, and T the temperature (°C). The nonlinear term in equation (2) was added to avoid a linear dynamical system for which the two possible asymptotic behaviors, either stability or instability, would have produced a mosquito's population vanishing or exponentially growing to infinite. So, in order to avoid useless evolutions, the auto-regulation term was introduced through a sort of larval carrying capacity K_L. In the model, this regulation is represented by the term $-m_L(T)\frac{L}{K_L}L$ which increases larval mortality as L increases. Regarding the last stage, with the introduction of the coefficient σ, it is assumed that A represents adult female mosquitoes only, the only ones that lay eggs and bite humans [11]. The influence of temperature on mosquito dynamics was modeled using the temperature dependent parameters reported in Table I, as suggested in [4], [5].

TABLE I: Temperature-dependent functions, [4], [5]

Expression and Definition
Transition rate from egg to larva [week^{-1}] $f_E(T) = (0.3 \times (1 + \cos(\frac{\pi(T-26)}{19}))) \times 7$
Transition rate from larva to pupa [week^{-1}] $f_L(T) = (0.3\exp[-((T-32.40)/10.20)^2]) \times 7$
Transition rate from pupa to adult [week^{-1}] $f_P(T) = (0.07\exp[-((T-36.29)/24.07)^2]) \times 7$
Egg mortality rate [week^{-1}] $m_E(T) = 0.12 \times 7$
Larval mortality rate [week^{-1}] $m_L(T) = (\exp(-T/2) + 0.2) \times 7$
Pupal mortality rate [week^{-1}] $m_P(T) = (\exp(-T/2) + 0.04) \times 7$
Adult mortality rate [week^{-1}] $m_A(T) = (\max\{0.05, 0.084 + 0.0032T\}) \times 7$
Oviposition rate per female mosquito [week^{-1}] $\beta(T) = (\max\{0, -0.016T^2 + 1.29T - 15.8\}) \times 7$

The larval autoregulation term given by $-\frac{m_L(T)}{K_L}L^2$ has been specifically introduced because this life stage is most sensitive to resource limitations, which strongly constrain survival.



(a) Eggs (b) Larvae

(c) Pupae (d) Adults

Fig. 2: Population dynamics of mosquito life stages during 2022–2024.

Figure 2 reports the population dynamics of the mosquito life stages during 2022–2024. A marked seasonal pattern can be observed, with recurrent increases and decreases

in population over time. Such an oscillatory behavior is consistent with the temperature-dependency of mosquitoes cycle of life, since temperature affects oviposition, maturation, and mortality phases. Eggs and adults show the largest amplitudes, whereas larvae and pupae present smoother variations, due to the intermediate nature of these stages within the mosquito life cycle. Moreover, it is worth to stress the prolonged duration of the peak in all the stages of mosquitoes in the period January-May 2024; this could be interpreted as a peculiarity in climate condition favouring mosquitos population increase. The consequences will be discussed in the numerical Section V.

B. Complete mathematical model

Taking into account the dynamics of the human population and introducing the dynamics of infected mosquitoes and infected humans, the following model is obtained, completed by the previously introduced equations, (1)–(4):

$$\dot{H} = cH(B-H) - dH \tag{5}$$
$$\dot{H}_I = \beta_{HM} A_I (H - H_I) - dH_I - \gamma H_I \tag{6}$$
$$\dot{A}_I = \beta_{MH}(A - A_I)H_I - m_A(T)A_I. \tag{7}$$

where H represents the human population, whose evolution is described by a logistic equation, H_I is the number of infected humans, and A_I represents the infected mosquitoes.

The meaning of the model parameters, along with the chosen numerical values, are reported in Table II [5], [12].

TABLE II: Model parameters

c	logistic growth rate (statistical data)	4×10^{-3} [week^{-1}]
B	carrying capacity (statistical data)	200×10^{6} [H]
K_L	larval carrying capacity	2.5×10^{8} [L]
d	natural mortality rate	10^{-6} [week^{-1}]
β_{HM}	transmission rate mosquito-human	to be estimated
β_{MH}	transmission rate human-mosquito	to be estimated
γ	recovery rate of infected humans	1 [week^{-1}]
σ	fraction of female mosquitos	0.5

In the model, β_{HM} represents the transmission rate from an infected mosquito to a healthy human, while β_{MH} denotes the transmission rate from an infected human to a healthy mosquito. Transmission rates are coefficients that include several factors, such as the probability of infection, the infective capacity of mosquitoes, the robustness of individuals upon contact with the virus, and so on; they will be identified in Section IV.

III. MODEL ANALYSIS

In this section, the proposed model is analyzed; firstly, the disease free equilibrium point (DFE) of the system is computed, and its stability is analyzed. Subsequently, the reproduction number is derived, showing the relationship with the stability property.

A. Disease-free equilibrium point

The determination of the equilibrium points is obtained by setting the state variation equal to zero, so getting

$$0 = \beta A - (m_E + f_E)E \tag{8}$$
$$0 = f_E E - \left[m_L(1+\tfrac{L}{K_L}) + f_L\right]L \tag{9}$$
$$0 = f_L L - \left[m_P + f_P\right]P \tag{10}$$
$$0 = \sigma f_P P - m_A A \tag{11}$$
$$0 = cH(B-H) - dH \tag{12}$$
$$0 = \beta_{HM} A_I (H - H_I) - (d+\gamma)H_I \tag{13}$$
$$0 = \beta_{MH}(A - A_I)H_I - m_A A_I \tag{14}$$

neglecting the explicit dependency from T for sake of compactness of the expressions. The Disease-Free Equilibrium point, denoted by the subscript DFE, is computed by assuming null the numbers of infected humans and mosquitoes, that is $H_I = H_{I,DFE} = 0$, $A_I = A_{I,DFE} = 0$. Then, equations (13) and (14) are always verified. From equation (12), along with the trivial solution of no human population ($H_{DFE} = 0$), one gets

$$H_{DFE} = B - \frac{d}{c} \tag{15}$$

feasible if

$$Bc - d > 0 \tag{16}$$

From (8), (11) and (10) one can write

$$E_{DFE} = \frac{\beta}{m_E + f_E} A_{DFE} = \alpha_1 A_{DFE} \tag{17}$$
$$P_{DFE} = \frac{m_A}{\sigma f_P} A_{DFE} = \alpha_2 A_{DFE} \tag{18}$$
$$L_{DFE} = \frac{m_P + f_P}{f_L}\frac{m_A}{\sigma f_P} A_{DFE} = \alpha_3\alpha_2 A_{DFE} \tag{19}$$

with evident meaning of the positive coefficients α_1, α_2 and α_3, thus expressing E_{DFE}, P_{DFE} and L_{DFE} as functions of A_{DFE}. By substituting (17) and (19) into (9), one obtains

$$f_E\alpha_1 A_{DFE} - (m_L + f_L)\alpha_3\alpha_2 A_{DFE} - \frac{m_L}{K_L}(\alpha_3\alpha_2 A_{DFE})^2 = 0 \tag{20}$$

from which the equilibrium value

$$A_{DFE} = \frac{K_L(f_E\alpha_1 - (m_L + f_L)\alpha_2\alpha_3)}{m_L\alpha_2^2\alpha_3^2} \tag{21}$$

is obtained along with the trivial condition of the absence of a mosquito population $A_{DFE} = 0$.

The solution (21) is feasible if and only if its non-negativeness condition is met. The equilibrium point is then

$$P_{DFE} = \begin{pmatrix} E_{DFE} \\ L_{DFE} \\ P_{DFE} \\ A_{DFE} \\ H_{DFE} \\ H_{I,DFE} \\ A_{I,DFE} \end{pmatrix} = \begin{pmatrix} \frac{\alpha_1 K_L(f_E\alpha_1 - (m_L+f_L)\alpha_2\alpha_3)}{m_L\alpha_2^2\alpha_3^2} \\ \frac{K_L(f_E\alpha_1 - (m_L+f_L)\alpha_2\alpha_3)}{m_L\alpha_2\alpha_3} \\ \frac{K_L(f_E\alpha_1 - (m_L+f_L)\alpha_2\alpha_3)}{m_L\alpha_2\alpha_3^2} \\ \frac{K_L(f_E\alpha_1 - (m_L+f_L)\alpha_2\alpha_3)}{m_L\alpha_2^2\alpha_3^2} \\ B - \frac{d}{c} \\ 0 \\ 0 \end{pmatrix} \tag{22}$$

B. Endemic equilibrium point

In this case, in order to determine the Endemic Equilibrium point, denoted by the subscript EE, notice that equations (8)–(12) do not depend on the infected individuals, neither human nor vectors; then, only the nonzero equilibrium values of H_I and A_I must be computed, solving equations (13) and (14), while the other components do not change. Starting from (13), one can write

$$A_I = \frac{(d+\gamma)H_I}{\beta_{HM}(H-H_I)} \tag{23}$$

which, once substituted into (14), yields to

$$\beta_{MH}AH_I - \frac{(d+\gamma)H_I}{\beta_{HM}(H-H_I)}(\beta_{MH}H_I + m_A) = 0 \tag{24}$$

Since $H_I = 0$ corresponds to the epidemic free condition, (24) can be simplified as

$$\beta_{MH}A - \frac{(d+\gamma)}{\beta_{HM}(H-H_I)}(\beta_{MH}H_I + m_A) = 0 \tag{25}$$

so getting

$$H_{I,EE} = \frac{\beta_{MH}\beta_{HM}H_{EE}A_{EE} - m_A(d+\gamma)}{\beta_{MH}(\beta_{HM}A_{EE} - (d+\gamma))} \tag{26}$$

Replacing (26) in (23), after computations one gets

$$A_{I,EE} = \frac{\beta_{MH}\beta_{HM}H_{EE}A_{EE} - m_A(d+\gamma)}{\beta_{HM}(m_A - \beta_{MH}H_{EE}))} \tag{27}$$

where the subscript EE denotes the endemic equilibrium point. The expressions of H_{EE}, E_{EE}, L_{EE}, P_{EE} and A_{EE} are the same as for the disease free conditions (15), (17), (19), (18) and (21) respectively.

Feasibility conditions require the non-negativeness of (26) and (27).

C. Stability analysis

In this section, the local stability of the disease-free equilibrium point is analyzed by applying the indirect Lyapunov criterion. Computing the Jacobian for the nonlieear dynamical system (1)–(7) one has

$$J = \begin{pmatrix} J_{11} & 0 & 0 \\ 0 & J_{22} & 0 \\ * & * & J_{33} \end{pmatrix} \tag{28}$$

with

$$J_{11} = \begin{pmatrix} -(m_E+f_E) & 0 & 0 & \beta \\ f_E & -(m_L+f_L)-\frac{2m_L}{K_L}L & 0 & 0 \\ 0 & f_L & -(m_P+f_P) & 0 \\ 0 & 0 & \sigma f_P & -m_A \end{pmatrix} \tag{29}$$

$$J_{22} = cB - d - 2cH \tag{30}$$

$$J_{33} = \begin{pmatrix} -\beta_{HM}A_I-(d+\gamma) & \beta_{HM}(H-H_I) \\ \beta_{MH}(A-A_I) & -\beta_{MH}H_I-m_A \end{pmatrix} \tag{31}$$

The structure of J puts in evidence that the model is composed by three subsystems.

The first is the insect dynamics (1)–(4), independent with respect to all the other variables and with the same equilibrium point for both the disease free and the endemic conditions, associated to the sub-matrix J_{11} evaluted at such a point, i.e. with L replaced with $L_{DFE} = L_{EE}$.

$$J_{11} = \begin{pmatrix} -(m_E+f_E) & 0 & 0 & \beta \\ f_E & -(m_L+f_L)-\frac{2m_L}{K_L}L & 0 & 0 \\ 0 & f_L & -(m_P+f_P) & 0 \\ 0 & 0 & \sigma f_P & -m_A \end{pmatrix} \tag{32}$$

Sufficient conditions about stability can be obtained by means of the Gershgorin theorem:

Theorem 1: The eigenvalues of a $n \times n$ matrix A with entries $a_{i,j}$ belong to the union of the n discs D_i, $i = 1,...,n$, where D_i is the disk centered in the diagonal elements $a_{i,i}$ and with radius equal to the sum of the moduli of the off-diagonal elements of the corresponding i-th column or the i–th row.

Since stability is given if and only if all the eigenvalues have negative real part, applying Theorem 1 by columns the following conditions assure that $Re(\lambda) < 0$:

$$-(m_E + f_E) + f_E < 0 \tag{33}$$

$$-(m_L + f_L) - 2\frac{m_L\alpha_2\alpha_3A_{DFE}}{K_L} + f_L < 0 \tag{34}$$

$$m_P + f_P + \sigma f_P < 0 \tag{35}$$

$$-m_A + \beta < 0 \tag{36}$$

The only nontrivial condition is (36), and this is the sufficient condition for stability of the vectors dynamics.

The second subsystem is given by human dynamics (5), with an equilibrium condition (15), that holds in both disease free and endemic conditions; the negativeness of the eigenvalue $\lambda_H = -(cB - d)$ is ensured by fulfilling the existence condition.

Finally, the third subsystem representing the infection transmission, given by (6) and (7), with different equilibrium points for the two conditions, disease free and endemic. To study their local stability, J_{33} must be considered, evaluated in the two equilibrium conditions respectively. For the disease free equilibrium point,

$$J_{33}|_{DFE} = \begin{pmatrix} -(d+\gamma) & \beta_{HM}H_{DFE} \\ \beta_{MH}A_{DFE} & -m_A \end{pmatrix} \tag{37}$$

and its eigenvalues, from the Routh–Hurwitz criterion, have negative real part if and only if

$$m_A(d+\gamma) - \beta_{HM}\beta_{MH}A_{DFE}H_{DFE} > 0 \tag{38}$$

while for the endemic equilibrium point

$$J_{33}|_{EE} = \begin{pmatrix} -\beta_{HM}A_{I,EE}-(d+\gamma) & \beta_{HM}(H_{EE}-H_{I,EE}) \\ \beta_{MH}(A_{EE}-A_{I,EE}) & -\beta_{MH}H_{I,EE}-m_A \end{pmatrix} \tag{39}$$

and the condition for negativeness of its eigenvalues, making use again of Routh–Hurwitz criterions and after some trivial computations, is

$$\beta_{HM}(m_A + \beta_{MH}H_{EE})A_{I,EE} + \beta_{MH}((d+\gamma) + \beta_{HM}A_{EE})H_{I,EE} + m_A(d+\gamma) - \beta_{HM}\beta_{MH}H_{EE}A_{EE} > 0 \tag{40}$$

D. Reproduction number

In epidemiology, the basic reproduction number R_0 is a useful parameter to provide an indication of whether an epidemic will expand or decline. In short, it estimates the number of susceptible individuals that the first infected subject in the population can infect in its infectivity period. If $R_0 > 1$, the number of infected individuals increases (and the larger R_0, the more extensive the contagion will be), while the epidemic tends to die out if $R_0 < 1$. For the calculation of the reproduction number, the next generation matrix approach proposed in [13] is adopted. Only the equations (6) and (7) corresponding to the infectious dynamics have to be considered.

They can be rewritten as

$$\begin{pmatrix} \dot{H}_I \\ \dot{A}_I \end{pmatrix} = \begin{pmatrix} \beta_{HM} A_I (H - H_I) \\ \beta_{MH} (A - A_I) H_I \end{pmatrix} - \begin{pmatrix} (d+\gamma) H_I \\ m_A A_I \end{pmatrix} = \zeta - \nu \tag{41}$$

To compute R_0, the next generation matrix K is used, where

$$K = \left(\frac{\partial \zeta}{\partial (H_I, A_I)} \Big|_{P_{\text{DFE}}} \right) \left(\frac{\partial \nu}{\partial (H_I, A_I)} \Big|_{P_{\text{DFE}}} \right)^{-1} = \begin{pmatrix} 0 & \dfrac{\beta_{HM} H}{m_A} \\ \dfrac{\beta_{MH} A}{d+\gamma} & 0 \end{pmatrix} \tag{42}$$

The eigenvalue with the largest modulus is the basic reproduction number, given by

$$R_0 = \sqrt{\frac{\beta_{HM} \beta_{MH} H_{DFE} A_{DFE}}{m_A (d+\gamma)}} \tag{43}$$

Since $R_0 < 1$ if and only if $R_0^2 < 1$, such a condition corresponds to $\beta_{HM}\beta_{MH}H_{DFE}A_{DFE} < m_A(d+\gamma)$. By considering the disease free epidemic stability conditions (36) - (38), it is deduced that if the disease free equilibrium point is stable then $R_0 < 1$, and viceversa, meaning that in this case the epidemic disease will not spread.

IV. IDENTIFICATION

Besides the model parameters introduced in Table II, it is necessary to estimate the transmission rates β_{HM}, from infected mosquitoes to healthy humans, and β_{MH}, from infected humans to healthy mosquitoes, from real data.

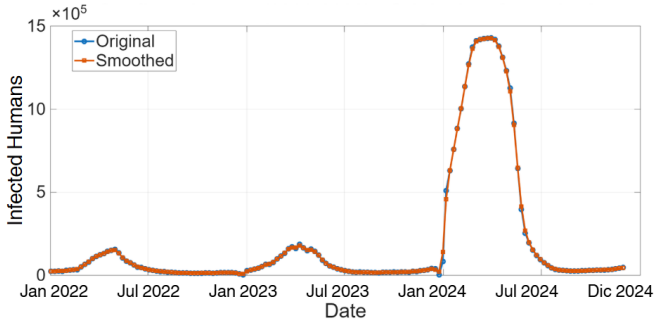


Fig. 3: Real and smoothed data.

For the identification procedure, official records of Dengue cases in Brazil during the years 2022–2023 were used as reference. The simulated trajectories produced by the proposed model were compared to the reported cases, after applying a Gaussian smoothing filter with a window size of 3 to the raw data to reduce short-term fluctuations, Fig. 3. This comparison provided the basis for calibrating the unknown parameters and assessing the model's ability to reproduce the observed epidemic dynamics. The chosen cost function is:

$$J(\beta_{HM}, \beta_{MH}) = \frac{1}{2} \int_{t_i}^{t_f} e^4 \, dt, \tag{44}$$

where t_i and t_f are the weeks from January 2022 to January 2023 respectively, and $e(t) = H_I(t) - I_R(t)$ is the error, with H_I the number of infected subjects estimated by the model and I_R the real data. The choice of a fourth-power error penalizes large deviations, forcing the identification to prioritize an accurate reconstruction of the peak magnitude and timing, which are the most epidemiological relevant features.

V. NUMERICAL RESULTS

In this section, the proposed model is studied numerically, adopting the parameters in Table II. The results of the identification procedure are reported in Figure 4. It can be observed that the proposed model is able to reproduce, with good accuracy, both the amplitude and the periodicity of the epidemic waves, which are clearly visible in the real data. The identified values of the transmission rates are $\beta_{HM} = 2.050 \cdot 10^{-12}$ and $\beta_{MH} = 1.937 \cdot 10^{3}$. This asymmetry between the two transmission rates is in line with previous studies on Dengue epidemiology, where the persistence of the disease is largely driven by the abundance of vectors rather than the presence of a small number of infected humans.

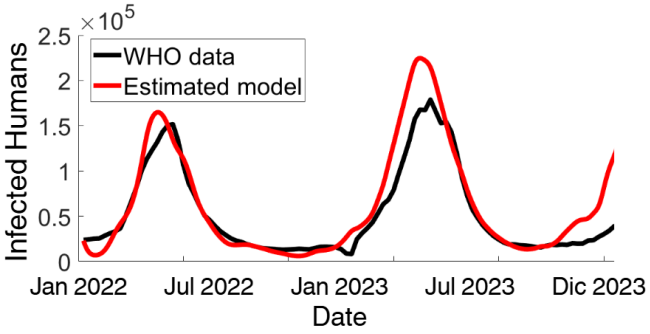


Fig. 4: Reconstruction of the number of infected humans.

Once that the unknown parameters have been estimated, it is possible to check numerically the stability conditions given in Section III taking into account the temperature dependencies. Then, taking a temperature range $T \in [20, 40]\,^{\circ}\text{C}$, the real parts of the eigenvalues of the Jacobian matrix are evaluated at the equilibrium points. The corresponding intervals are reported in Table III for the disease-free equilibrium and in Table IV for the endemic equilibrium.

For the disease-free equilibrium, the spectrum always contains at least one positive eigenvalue for all the considered temperature range. This indicates that the disease-free equilibrium point is locally unstable for the considered numerical case. At the same time, for the endemic equilibrium

TABLE III: Intervals of the real parts of the Jacobian eigenvalues at the disease-free equilibria for $T \in [20, 40]\,^{\circ}\mathrm{C}$.

Eigenvalue	DFE ($H = B - \frac{d}{c}$)	DFE ($H = 0$)
$Re(\lambda_1)$	$[-5.55 \cdot 10^8, -3.87 \cdot 10^7]$	$[-5.55 \cdot 10^8, -3.87 \cdot 10^7]$
$Re(\lambda_2)$	$[-1.41 \cdot 10^8, -1.83 \cdot 10^7]$	$[-5.04, -1.52]$
$Re(\lambda_3)$	$[-8.0 \cdot 10^5, -8.0 \cdot 10^5]$	$[-1.48, -1.03]$
$Re(\lambda_4)$	$[-5.04, -1.52]$	$[-1.48, -1.03]$
$Re(\lambda_5)$	$[-1.48, -1.03]$	$[-1, -1]$
$Re(\lambda_6)$	$[-0.77, -0.59]$	$[-0.77, -0.59]$
$Re(\lambda_7)$	$[1.83 \cdot 10^7, 1.41 \cdot 10^8]$	$[8.0 \cdot 10^5, 8.0 \cdot 10^5]$

TABLE IV: Intervals of the real parts of the Jacobian eigenvalues at the endemic equilibrium for $T \in [20, 40]\,^{\circ}\mathrm{C}$.

Eigenvalue	Endemic
$Re(\lambda_1)$	$[-3.87 \cdot 10^{11}, -3.87 \cdot 10^{11}]$
$Re(\lambda_2)$	$[-5.55 \cdot 10^8, -3.87 \cdot 10^7]$
$Re(\lambda_3)$	$[-8.0 \cdot 10^5, -8.0 \cdot 10^5]$
$Re(\lambda_4)$	$[-5.16 \cdot 10^4, -8.69 \cdot 10^2]$
$Re(\lambda_5)$	$[-5.04, -1.52]$
$Re(\lambda_6)$	$[-1.48, -1.03]$
$Re(\lambda_7)$	$[-0.77, -0.59]$

all eigenvalues exhibit negative real parts, implying local asymptotic stability and therefore persistence of the infection once established.

Finally, to exploit the prediction capability of the proposed model, in Figure 5 the simulated time evolution of the infected human population over the period 2022–2024 is superimposed to the real data. The latter shows an abnormal peak corresponding to half 2024. The model partially captures such an extreme amplitude, showing the contribution of temperature variations with consequent increment of mosquitos number, evidenced in Figure 2 by the longer time of high number of insects.

Besides this modeled effect, the marked discrepancy could be partly explained by additional environmental drivers not included in the present model. Moreover, according to Brasilian Government [14], it must also be considered that between 2023 and 2024, facing the dangerous increase of cases, the Brazilian government devoted higher investments to address the health emergency by more than 50%, significantly strengthening testing and laboratory surveillance. This led to an increase in diagnoses of over 76.4%.

VI. CONCLUSIONS

In this study, a compartmental model was formulated and analyzed to describe the transmission dynamics of the Dengue virus under the explicit influence of temperature on mosquitoes development. Real data of Dengue cases reported in Brazil between 2022 and 2023 were used for identifying the contact rate parameters; the results show that the model is able to reproduce the observed dynamics of Dengue cases, successfully capturing both the number of infections and the seasonally oscillatory trend of the epidemic. The possible use of the considered model for sanitary emergency prediction was partially successful, being able to capture the higher number of infected individuals, at least for the contribution of the temperature, and then of number of mosquitoes. On the other hand, for the real data, it is evident that there have been additional contributions, represented by human interventions, not here modeled but to be included in an ongoing work.

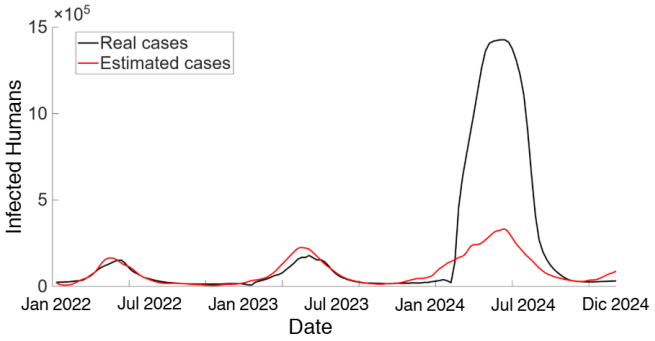


Fig. 5: Comparison between real cases and estimated cases from 2022 and 2024.

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